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Environmental Radiation and the Lung

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Environmental sources of radioactive materials and their relation to lung doses and lung burdens are described. The approaches used and the problems encountered in estimating lung doses are illustrated. Exposure to radon daughter products is contrasted to exposure to plutonium as particular examples of the hazards associated with radioactive materials of different chemical and physical characteristics.

Introduction

The radiation dose received by man from the many radioactive sources present in his environment may be thought of for categorical simplicity as due to either external or internal radiation. This simplification is always complicated by the various routes of transfer of radioisotopes from the exterior to the interior of man. Radioactive materials may be ingested through the mouth, inhaled into the lungs, or absorbed through the skin and breaks in the skin.

The lung is unique in that it can receive radiation from internally deposited material while at the same time being continually exposed to air from the external environment which may also contain radioactive material. For exposure to many radioisotopes the lung is the critical orgran. The International Commission on Radiation Protection (ICRP)*(1)* has recognized the lung and the gastrointestinal tract as critical organs for most insoluble forms of the radionuclides. Solubility is only one of several factors which affect clearance from the lung and its choice as the critical organ. Some of the other factors will be discussed later.

In this paper we will consider those radioisotopes in the natural environment as well as some produced by man's activity to which large segments of the world population may be exposed and for which lung exposures should be considered. Dose calculations will be reviewed and compared for two selected cases, radon daughters and plutonium.

Naturally Occurring Materials as Sources of Radiation Exposure

Any assessment of the hazard associated with a certain level of radiation must be made from the results of the many experimental studies that have been made of the biological effects of radiation. When this is done, it is apparent that most experiments which correlate definite biological effects with radiation dose are at high radiation levels with respect to background or natural radiation levels. Although it is difficult to extrapolate to levels slightly above the natural baseline, the average exposure man receives from natural radiation serves as a baseline from which the effects of additional exposure may be judged *(2)*.

It is assumed as a general philosophy in radiation protection that any radiation level is potentially hazardous even at or below the average levels of natural radiation. Levels due to natural radiation at any particular locality may differ from this average by orders of magnitude. Even small increases in exposure over baseline levels may be important when considering effects on large populations or on future populations.

The lung is directly exposed to naturally oc-

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curring radioactive materials through inspired air, and it is important to examine the sources of natural radiation and to consider the possible means and consequences of exposure to naturally occurring airborne radionuclides.

Natural radiation may be divided into two categories: that produced by the radioactive elements that have been present in the upper crust of the earth for millions of years (primordial), and cosmic radiation that bombards the earth each day and also creates new radioactive materials.

It is estimated that the total average whole-body dose from natural radiation in the United States due to terrestial radiation is 85 mrem/yr and that due to cosmic radiation is 45 mrem/yr. Of the 85 mrem/yr terrestial radiation, 25 mrem/yr is estimated to be from internal radiation *(3a)*. In this report, we shall be primarily concerned with the internal dose to the lung from inhaled radionuclides. Factors that determine these doses will be discussed in the last section.

The most important radioactive elements in the earth's crust contributing to man's dose from natural radiation are the isotope ^{40}K and isotopes of the uranium and thorium series. The actinium series and other primordial radioisotopes in the earth's crust contribute very little to man's radiation dose *(4–9)*. The isotope ^{40}K contributes about 17 mrem/yr of the 25 mrem/yr average internal whole body dose *(3b)*.

Isotopes of the uranium and thorium series are listed in Table 1. Uranium occurs in the earth's crust at a concentration of about 3–4 ppm and thorium at 11–15 ppm *(9)*. By contrast, the actinium series starts with ^{235}U (less than 1% of U or less than 0.03 ppm) and therefore the percentage abundance of isotopes in the actinium series is small. Many of the other primordial isotopes have long half-lives resulting in low specific activity (the specific activity of an isotope depends inversely on the half-life). For example ^{138}La has a half-life of 1.2×10^{11} yr. which results in a specific activity of 2.3×10^{-2} μCi/g. The abundance of ^{138}La is 0.089% (the rest is stable ^{139}La) so that even if pure lanthanum were obtained, the specific activity would be only 2.05×10^{-5} μCi. If 50 μCi is taken as the (maximum permissble body burden) (MPBB), then 24.4×10^2 kg of lanthanum would have to be inhaled or ingested to represent a MPBB. Although the above calculation is for a specific isotope, it does clearly show the reduced hazard that is associated with very long-lived isotopes.

If we make a somewhat arbitary cutoff at a half-life of 10^{11} yr, then the only other primordial radionuclides left for consideration are ^{187}Re, ^{176}Lu, and ^{87}Rb. The concentrations of ^{187}Re (0.001 ppm) and ^{176}Lu (0.01 ppm) in the earth's crust are small compared to those of uranium and thorium *(9)*. Rubidium-87 (75 ppm)

Table 1. Isotopes of the uranium and thorium series.

Uranium series		*Thorium series*	
Isotope	Half-life	Isotope	Half-life [a]
U–238	4.51×10^9 yr	Th–232	1.47×10^{10} yr
Th–234	24.10 day	Ra–228	6.7 yr
Pa–234m	1.18 min	Ac–228	6.13 hr
Pa–234	6.7 hr	Th–228	1.91 yr
U–234	2.47×10^5 yr	Ra–224	3.64 day
Th–230	8.0×10^4 yr	Rn–220	55.3 sec
Ra–226	1.62×10^3 yr	Po–216	0.145 sec
Rn–222	3.82 day	Pb–212	10.6 hr
Po–218	3.05 min	At–216	3×10^{-4} sec
Pb–214	26.8 min	Bi–212	60.6 min
At–218	1.5–2 sec	Po–212	3.04×10^{-7} sec
Bi–214	19.7 min	Tl–208	3.10 min
Po–214	1.64×10^{-4} sec	Pb–208	Stable
Tl–210	1.32 min		
Pb–210	20.4 yr		
Bi–210	5.01 day		
Po–210	138 day		
Tl–206	4.19 min		
Pb–206	Stable		

[a] Half-lives obtained from *Radiological Health Handbook (10)*.

is the most abundant of the natural radioactive nuclei but has a long half-life (5×10^{10} yr) and has a single beta emission (0.024 Mev) so that the expected dose is small. It is estimated as 0.6 mrem/yr whole body *(3b)*.

Radon and Daughters

Those isotopes listed in Table 1 which become airborne are of particular interest. Obviously the radioactive gases and their decay products must receive primary consideration. In trying to determine those isotopes which potentially would be of more harm to the lung, it is recognized that almost any isotope can become airborne either as a gas or on dust particles and deliver doses of radiation to the lung. The uranium and thorium series both contain gaseous isotopes of radon. Radon-222 of the uranium series has a half-life of 3.82 days and has a large number of daughter products, some with long half-lives. For example, ^{210}Pb has a half-life of 20.4 years, ^{210}Po of 138 days, and ^{210}Bi of 5 days. In the thorium series, ^{220}Rn occurs with a half-life of 55.3 sec. The longest lived daughter product of ^{220}Rn is ^{212}Bi with a half-life of 60.6 min. The actinium series also has a radon isotope ^{219}Rn with a half-life of 4.0 sec and the longest-lived daughter product is ^{211}Pb with a half-life of 36.1 min. Therefore, the thorium and actinium series will contribute very little to man's lung dose from gaseous products except for strictly local conditions where high concentrations or uranium and thorium exist (as in mines). Even here the principal gas for consideration will be ^{222}Rn. Measured concentrations at various locations show ^{222}Rn to be 50 to 100 times more concentrated than ^{220} Rn *(5b)*. Parent isotopes of radon in the uranium and thorium series contribute to the lung dose through inhaled radioactive dust containing ^{238}U, ^{234}Th, ^{234}Pa, ^{234}U, ^{230}Th, ^{226}Ra, ^{232}Th, ^{228}Ra, ^{228}Ac, ^{228}Th, and ^{224}Ra. The estimated average annual internal whole-body radiation dose due to ^{222}Rn is 3 mrem/yr *(3b)*. However, the estimates to the lung are greater. These estimates will be discussed later.

Atmospheric concentration of radon depends on many geological and meteorlogical factors. Such factors as proximity to uranium and thorium ore deposits, porosity of the soil, effect of temperature on diffusion rates, snow cover to prevent the escape of radon gas from the ground, and wind conditions play an important role in the concentrations observed for radon. Osburn *(11)* considers migration of radon out of soil where it is formed to occur in four steps: recoil of radon precursors, diffusion through the mineral grain, movement through permeable rock or soil and release to the environment. Depending on soil surface, much of the gas may be absorbed. For example, if charcoal is present as in the case of ground covered with a burned forest, then much gas is absorbed. Since radon gas is heavy, it also tends to accumulate in valleys. Rain may cause daughter products to return to ground and increase ground radiation temporarily; in other cases wet ground may prevent the escape of the gas and thus reduce ground radiation.

When ^{222}Rn decays with emission of an alpha particle (5.47 MeV), the newly formed atom (^{218}Po) has a recoil energy of 0.11 MeV which is enough to give most atoms a positive charge *(12)*. These atoms may later become neutralized by electrons or may attach to aerosol particles from dust, smoke, ocean spray or pollen. Gold et al. *(13)* find that the major fractions of radon daughters are associated with particles of 0.005 to 0.04 μm size. Others *(5c)* find most of the activity on 0.2–0.084 μm size particles. Raabe *(14)* finds that the attachment of activity to an aerosol particle is proportional to the surface area of the particles for all particle sizes except at extremely high radon concentrations.

Gold, et al. *(13)*, in a 1953 Cincinnati study, found the average annual ^{222}Rn concentration to be 260 pCi/m^3. In this same study, the concentration of ^{7}Be was found to be 0.096 pCi/m^3 and of ^{210}Pb to be 0.0082 pCi/m^3. Lockhart *(15)* reports ^{222}Rn concentrations studies for various sites showing concentration values of 0.47 pCi/m^3 at the South Pole up to 122 pCi/m^3 in Washington, D.C. In Czechoslovakia, ground level values for ^{222}Rn were observed *(16)* from 26 pCi/m^3 to 106 pCi/m^3. Shearer *(17)* has evalated ^{222}Rn concentration near uranium mine tailings in Colorado and Utah. At Grand Junction, Colorado, the level recorded was 800 pCi/m^3, whereas directly over the tailings, levels of 3.5×10^3 to 1.6×10^4 pCi/m^3 were observed (ICRP recommendation for the maximum permissible concentrations for the general population is 10^3 pCi/m^3).

The hazard of naturally occurring radioactive materials to the lungs of the general population therefore reduces to much the same hazard as for uranium miners, except exposure is less per individual but to a greater number of individuals. The problem or uranium miners has been extensively studied and many lung models and dosimetry calculations have been made in connection with the problem encountered in uranium mining. These will be discussed briefly in the section on dose calculations for radon and radon daughters.

Cosmic Radiation

Cosmic radiation is radiation which comes from outer space and is incident on the earth's atmosphere where numerous nuclides are produced when these rays interact with the gaseous elements in the troposphere. For example, ^{14}C is produced by the reaction of cosmic neutrons on nitrogen ($^{14}N+n \rightarrow {}^{14}C+H$). ^{7}Be and ^{10}Be are spallation products of nitrogen. ^{7}Be is easily detected in the atmospheric air and is precipated with rain. ^{10}Be is observed in deep sea cores. Reactions with argon in the upper atmosphere produces ^{22}Na, ^{32}Li, ^{33}P, ^{35}S, ^{36}Cl, and ^{39}Cl. Spallation of nitrogen and oxygen produces tritium. In addition to these isotopes, several others may be produced by spallation reactions. Perkins *(18)* lists 14 different isotopes and indicates most of these are spallation products. Bartels *(19)* suggests as an alternate hypothesis that volcanic action around the world contributes particles of various elements to the atmosphere and that the primary method of production is by neutron activation of these volcanically produced particles. As noted previously, cosmic radiation has been estimated to contribute 45 mrem/yr of the natural external radiation dose. The dose estimate for the average yearly internal dose to the whole body from ^{14}C is 1 mrem/yr and from ^{3}H is 0.004 mrem/yr *(3b)*. Dose rates due to other isotopes mentioned above are small. Carbon and hydrogen are integral parts of most biological molecules so that ^{14}C and ^{3}H will deliver radiation doses to the whole body (the dose due to ^{14}C is the only significant dose). Therefore, the whole body dose rather than dose to the lung is the primary consideration for these isotopes.

Man-Made Sources of Radiation Exposure

Radiation hazards which are greatest but which can be controlled are those which are created by various activities of man. Although natural radioactivity is widespread, it does not exist to a degree that deleterious effects on man can be unequivocally established except in a few localized cases (as with uranium miners). Man's activity not only produces localized hazards but may produce hazards which are almost as widespread as natural radiation.

Nuclear Weapons

Radiation produced by fallout from atomic weapons is the principle source of widespread radiation exposure produced by man to date. Fallout produced by the detonation of nuclear explosives may be divided into three categories: local, intermediate, and worldwide *(20)*. Local fallout is fallout deposited within the first 24 hr after the explosion and confined to the immediate vicinity of detonation (may extend several hundred miles under windy conditions).

Intermediate fallout occurs within a few days to a month and consists of fission products deposited in the troposphere (the atmosphere from the surface of the earth up to 35,000 to 55,000 ft). Essentially all this fallout occurs in a poorly defined band around the world in the same general latitude in which the explosion occurred. Although confined to the same general latitude, fallout from any one explosion is spotty with greatest fallout occurring in areas of precipitation.

Worldwide fallout occurs over time periods of several months to several years and is due to deposition of fission products in the stratosphere. Fallout from stratospheric fission products is widespread over the earth but is generally restricted to the same hemisphere as the detonation. Stratospheric injection is almost complete for nuclear devices greater than 500 kilotons *(5d)*.

The amount of fallout in each of the above three categories depends on many factors. Of prime importance is energy release of the weapon and its position with respect to the land or water surface at the time of detonation. Fission products will be deposited in the stratosphere only for higher yield weapons. For

weapons in the kiloton range all the fallout will be restricted to local and intermediate deposition. However, over 90% of all fission products produced by the detonation of nuclear devices resulted from 1 megaton or greater weapons *(21)*. Some weapons are detonated underground resulting in very little activity above ground. Others detonated underground may produce craters and some local and intermediate fallout. Accidental venting also leads to the injection of volatile fission products into the atmosphere.

The fission products produced by a nuclear explosion consists of over 200 different nuclides of about 36 elements between atomic number 28 and 65 *(22)*. Other radioactive elements may be produced by neutron activation of elements in the ground, air, or structural parts of the nuclear device. The relative amounts of isotopes produced depends on the type nuclear device and its surroundings when detonated.

In 1958, a voluntary moratorium on nuclear weapon testing was agreed to by the United States, the United Kingdom and the Soviet Union. In 1960, France exploded three nuclear devices, and in 1961, the Soviet Union resumed testing. The United States followed with a series of tests in 1962. A monitoring station in Cincinnati reported a maximum activity (gross beta) of 74 pCi/m^3 on November 27, 1962 for that city *(23)*. Since 1962, only a few atmospheric tests by the French and Chinese have been conducted and the level of activity has decreased.

The French and Chinese are continuing their atmospheric testing with the most recent detonation at the time of this writing occurring in the spring and summer of 1973.

Of the many radionuclides produced by a detonation, those with relatively long half-lives will be the most hazardous for the general population under testing conditions (wartime conditions will not be considered). The principal isotopes of concern for whole body dose are ^{90}Sr and ^{137}Cs with half lives of 28 and 30 yr, respectively. These are important biologically, because ^{90}Sr is chemically similar to calcium and ^{137}Cs to potassium. Whole body per capita U.S. doses estimated for 1963, 1965 and 1969 are 13, 6.9, and 4 mrem/yr, respectively *(3c)*. By the end of 1961, naturally occurring ^{14}C had been increased by 1.41% *(6)*. In addition to ^{90}Sr and ^{137}Cs, principal isotopes contributing to lung dose are ^{95}Zr, ^{144}Ce, ^{238}Pu, and ^{239}Pu. For most radionuclides such as ^{89}Sr, ^{137}Cs, ^{131}I, ^{14}C, and ^{3}H, the effective half-life in man is less than 100 days, and 90% or more of the dose is received 1 yr after exposure *(24)*. But for isotopes such as ^{90}Sr which is deposited in the bone and ^{239}Pu which is essentially insoluble and is deposited in the lung, resulting in long biological as well as physical half-lives, an individual will continue receiving radiation for the remainder of his life.

Shleien *(25)* estimates that at least 88% of the fallout activity is associated with particles less than 1.75 μm in diameter and that the mean diameter is 0.38 μm. Fresh fallout is usually associated with particles of larger diameter. Of the activity from the fallout, 70% is associated with particles of similar geometric mean diameter as airborne dust in the area.

The activity of individual particles has been determined showing that the fraction of particles with activities equal to or greater than 0.01 pCi decreased from 66.3% in November 1962 to only 8.5% in March, 1963 *(26)*. The concentration of particles with activities less than 0.01 pCi increased from 2.2×10^4 to 3×10^4/m^3 during the same period. The average activity per particle in November 1962 was 5×10^{-4} pCi and in March 1963 was 2×10^{-4} pCi.

The U.S. per capita doses to the lung from inhalation of fallout for the years 1963, 1965, and 1969 are estimated as 17, 1.6, and 0.6 mrem/yr, respectively *(6)*. When the site of deposition is taken into account, the dose estimates to that site are considerably increased. This will be discussed later in more detail when plutonium and radon daughters are treated.

Power Plants

Another source of manmade radiation which is more localized but which will become more widespread is that associated with the nuclear power industry. This includes not only the effluents from the power producing reactors but also the products released from fuel manufacturing and reprocessing plants.

A reactor accumulates large quantities of fission products but only a small percentage of these are released to the environment. Reactors generally employ a closed primary coolant loop so that most of the fission products which leak into the coolant and the neutron-activated

materials in the coolant and reactor structural materials are contained within the reactor. There are two exceptions in the U.S.: the air-cooled graphite moderated reactors at Oak Ridge and Brookhaven and the water-cooled plutonium-producting reactor at Hanford, Washington *(5e)*. In the United Kingdom, there are also several gas cooled reactors which use CO_2 as the coolant. ^{41}A and ^{14}C, along with small amounts of fission products are released to the air from these reactors. ^{41}A has a short half-life (1.8 hr) so there is no problem with build up of activity in the environment. For example, at Brookhaven up to 750 Ci of ^{41}A/hr are released from the 400-ft stack but at the perimeter of the site the concentration has been judged to be of little concern *(5e)*.

All reactors release some of the noble gases to the environment along with some volatile fission products such as iodine, cesium and oxides of tellurium and rubidium. Present reactor operations remove most of these products or hold them in charcoal beds or tanks long enough for many of the radioisotopes to decay to low levels. For example, ^{133}Xe and ^{85}Kr are two noble gases which are usually retained in charcoal beds before release to the environment. With a half-life of 5.3 days, ^{133}Xe is considerably reduced in activity by the holdup, but the ^{85}Kr ($T½$ = 10.8 yr) is little affected. This has led to some concern about buildup of ^{85}Kr in the environment.

Martin et al. *(27)* have compared activity released from nuclear plants and from fossil fuel plants (naturally occurring radionuclides are in fossil fuels) and found that in some cases a coal-burning plant may release more activity than an electrically equivalent nuclear plant. Since fossil fuels contain primordial radioisotopes, it is conceivable that if the emission from fossil fuel plants are not closely controlled that the dose to the lung from the inhalation of insoluble particles could be equal or greater than that from a carefully controlled nuclear plant.

A greater hazard than nuclear plants except under accident conditions is that associated with fuel reprocessing plants. Here fuel and fuel cladding containing enormous amounts of fission products are dissolved and reprocessed to retrieve unburned ^{235}U and ^{238}U and to remove formed ^{239}Pu. For example, a 500 MW reactor which has been in operation for 180 days has a total activity of 4.1×10^8Ci with 8.4×10^7Ci of this being represented by volatiles. Iodine represents 5×10^7Ci and noble gases 3.4×10^7Ci *(5f)*. Of major concern are the fission products with long half-lives, since the fuel is normally stored several months to allow decay of the products with short half-lives. Essentially all of ^{85}Kr produced (0.3% fission yield) may be released to the environment.

The concentration of ^{85}Kr in 1969 was estimated as 33 dpm/m^3 or 15 pCi/m^3 and increasing linearly at about 1 pCi/m^3/yr *(28)*. Hendrickson *(29)* has estimated the dose from ^{85}Kr to the skin, whole body and lung for immersion in a cloud of ^{85}Kr of concentration 3×10^5 pCi/m^3, equal to 1/10 the maximum permissible body burden for occupational workers given by the ICRP. He estimates this concentration will be reached by the year 2050 if all ^{85}Kr that is produced is released to the environment. He finds this level to give a dose to the lung of 12 mrem/yr. Diethron *(30)* had estimated the dose from ^{85}Kr in 1968 to be 3×10^{-3} mrad/yr to the bare skin and respiratory system. Methods are under development to retain ^{85}Kr in permanent storage either in pressurized tanks, charcoal beds at low temperature, or by entrapment in molecular cage like compounds.

In addition to ^{85}Kr, fuel reprocessing plants will also release ^3H as well as other fission products. Tadmor *(31)* estimates that a 10 ton/-day reprocessing plant will release 2.7×10^{-2} Ci/sec of ^3H and 1 Ci/sec of ^{85}Kr. Much of the ^3H will be in the form of water vapor and will become mixed with the total water environment, whereas ^{85}Kr, which is not very soluble, will remain in the gaseous state.

Under maximum credible accident conditions it is usually assumed that all volatiles and 1–10% nonvolatiles will escape to the atmosphere *(5e)*. From the analysis of the few accidental releases of fission products from reactors which have occurred, it appears that the principal short-term hazard is associated with release of ^{131}I. Iodine is concentrated in the food chain, milk, and in the human thyroid, the dose by ingestion being much greater than the dose by inhalation. Many potential problems with reactors are related to overheating of the core and possible meltdown or burning. An accident

at Windscale in England is a good example of this. During an annealing operation, the reactor overheated and the uranium metal fuel caught fire, resulting in the release of many fission products. Since this experience, uranium fuel is now usually made of UO_2 which is not as likely to burn. However, any time there is a fire at a reactor, fuel production facility, fuel reprocessing facility, or of any stored radioactive material there is an increased danger of releasing material into the atmosphere. The United Kingdom Atomic Energy Commission has given as a safety guide that a fraction of 10^{-6} for metals of high melting point may be aerosolized. For volatile pyrophoric compounds the fraction may be as high as 10^{-3}. A fraction of 10^{-4} is used for intermediate materials. Where small amounts are in storage, this fraction may be multiplied by the amount in storage. If the result is less than the ICRP limits for maximum permissible dose then it can be assumed there is little danger from radiation even if a fire occurs *(32)*.

Other Uses

Other uses of radioisotopes are increasing such as the use of radionuclides as heat sources for small power generators. These heat sources require from 27.2 to 2700 Ci/W, so that for any appreciable power, large quantities of activity are required *(5g)*. Isotopes which have been used are ^{210}Po, ^{242}Cm, ^{238}Pu, ^{144}Ce, ^{147}Pm, ^{137}Cs, and ^{90}Sr. Burnup of these sources such as occurred with a ^{238}Pu loaded satellite *(24)* will lead to aerosolization of these extremely hazardous materials. Cardiac pacemakers and other equipment which may be powered by these sources must be very rugged to ensure no leakage of these dangerous isotopes.

All possible uses (such as medical) of radioisotopes and their associated lung hazards are too numerous to mention and could not be adequately discussed in a paper of this nature. Animal experimental studies, epidemiological studies on humans, and theoretical dose calculations are discussed in the following sections.

In the foregoing sections, an attempt was made to identify those isotopes which are the most important as possible contributors to lung dose. In the natural environment, radon and its daughter products appear to be those of most concern. Table 3 lists some of the relevant physical properties. For exposure to isotopes produced by the activity of man, the process of selecting the most hazardous isotopes is more complicated. However, based on our previous discussions, the isotopes listed in Table 2 along with some of their physical properties are isotopes which may be expected to become of more concern as nuclear uses expand. This list is certainly not exhaustive and may omit isotopes that will become more widespread as new technologies are introduced. Rather than review the pertinent dose calculations for each of the isotopes listed, we have selected two cases to il-

Table 2. Physical characteristics of selected radioisotopes and their principal daughters.[a]

Isotope	Half-life[a]	Major radiations	Energy, Mev [b]
Kr-85	10.8 yr	β-	0.67
Sr-90	27.7 yr	β-	0.546
Y-90	64 hr	β-	2.27
Zr-95	65.5 day	β-	0.89(2%), 0.396(55%) 0.36(43%) 0.723(49%), 0.756 (49%)
Nb-95	35 day	β-	0.16 0.765
Cs-137	30 yr	β-	1.176(6.5%), 0.514 (93.5%) 0.662 (85%)
Ce-144	284 day	β-	0.31(76%), 0.175 (24%)
Nd-144	17.3 min	β-	3.0(97.8%) 0.695(1.5%), 1.487 (0.29%), 2.186(0.7%)
Pm-147	2.62 yr	β-	0.224
Po-210	138.4 day	α	5.305
Pu-238	86.4 yr	α	5.5(72%), 5.46(28%)
Pu-239	24,390 yr	α	5.16(88%), 5.11(11%)
Cm-242	162.5 day	α	6.12(74%), 6.07(26%)

[a] From *Radiological Health Handbook (10)*.
[b] Beta energy given is maximum beta energy.

Table 3. Radon and radon daughters.

Isotopes	Half-life [a]	Major radiations	For α-emitting isotopes	
			Energy, MeV	Tissue range, μm[b]
Rn-222	3.82 day	α	5.49	41
Po-218 (Ra A)	3.05 min	α	6.00	47
Pb-214 (Ra B)	26.8 min	β-		
Bi-214 (Ra C)	19.7 min	β-		
Po-214 (Ra C')	1.64×10^{-4} sec	α	7.69	71
Pb-210 (Ra D)	20.4 yr	β-		
Bi-210 (Ra E)	5.01 day	β-		
Po-210 (Ra F)	138 day	α	5.31	39
Pb-206 (Ra G)	Stable			

[a] From *Radiological Health Handbook (10).*
[b] Data of Lea *(37).*

lustrate how dose calculations have been made for the lung and the problems encountered in obtaining meaningful dose estimates. The isotopes we wish to consider are radon daughters and plutonium. Radon daughters were chosen because of their widespread occurrence in the environment and because considerable work has been done in determining the dose to lung from radon daughters particularly as relates to uranium miners. Plutonium was chosen because of its high toxicity and current interest.

Respiratory Tract Deposition: Radon Daughters

Historical Summary

Several authors have considered the dose to the respiratory tract from radon and its daughters in considerable detail *(33–36).* Since this has already been done we will not repeat the detailed calculations or give a lengthy review but will present those facts which we feel are applicable for an appreciation of the principles used for the determination of dose and their limitations. The decay scheme for radon is given in Table 3. The polonium isotopes are alpha emitters and account for the major portion of the dose to man's respiratory tract. Men engaged in several types of underground mining have experienced exposure to radon decay products which in some instances greatly exceed exposure to the general population. Excess respiratory tract cancer has been found among U.S. and European uranium miners *(35,38–40).* A major cause of excess cancer was the inhalation of radon daughters *(41).* Many attempts have been made to determine the dose to uranium miners from these radon daughters and we will review the results of those attempts here.

Parker *(42),* Walsh *(33),* and Nelson et al. *(34)* have reviewed the published dose calculations for uranium miners. We will give here essentially a synopsis and update of the review by Walsh *(33).*

During a study in uranium mines, the U.S. Public Health Service developed the working level (WL) exposure concept *(36).* One WL was defined as any combination of radon daughters in one liter of air which will result in the emission of 1.3×10^5 Mev of alpha energy in the complete decay through RaC'. Cumulative exposure is given in working level months (WLM). One WLM is defined as an exposure to a concentration of one WL for 170 hr. This exposure definition recognized the major contribution to the dose from the alpha-emitting radon daughters (RaA and RaC', Table 3). One working level is associated with 100 pCi/l. of radon at equilibrium with its daughters *(43).*

Holleman *(44),* in 1968, reviewed the published dose calculations through 1967. In Table 4 is given his summary as well as the results of more recent calculations. Morgan *(45)* in testimony before the Joint Committee on Atomic Energy in 1967 reviewed the biology of the setting of maximum permissible exposure limits for mine workers and the problem in choosing the most appropriate dose model and critical tissue. As can be seen in Table 4, results have been at variance. One of the obvious reasons for this variance is choice of critical tissue. The calculations of Altshuler *(46),* Jacobi *(47),* Haque and Collinson *(48),* and Harley *(49)* refer to the highest dose rate to a particular

Table 4. Summary of published dose calculations for radon and radon daughters.[a]

Investigator	Isotope contributing to dose	Critical tissue	Calculated dose, rad/WLM
Evans and Goodman *(50)*	Radon only	Lung	0.002
Lorenz *(51)*	Radon only	Lung	0.002
Mitchell *(52)*	Radon only	Bronchi	0.01
Shapiro *(53)*	Radon plus daughters	Tertiary bronchioles	0.6
Chamberlain and Dyson *(54)*	Radon plus Ra A only		0.4
Holaday et al. *(36)*	Radon plus daughters	Main bronchi, bronchial tissue	2.3
Thomas *(55)*	Ra A only	Bronchial tissue	<0.1
Altshuler, Nelson, and Kuschner *(46)*	Daughters	Segmented bronchi	3.0
Jacobi *(47)*	Daughters	Secondary-quaternary bronchioles	2.9
Haque and Collinson *(48)*	Daughters	Segmented bronchi	12.0
Harley and Pasternak *(49)*	Daughters	Segmented bronchi	0.2–0.3
Walsh *(33)*	Daughters	Bronchial epithelium	<1.0

[a] Daughters imply short-lived daughters only (Ra A, Ra B, Ra C, and Ra C').

region, while the calculation of Walsh *(33)* refers to the average dose to the bronchial epithelium of the tracheobronchial tree. The other reasons for the variance relate to the assumptions regarding aerosol characteristics, deposition calculations including respiratory tract model adopted and methods of depth-dose calculation. Hereafter, the discussion will be limited to those calculations which considered radon daughters.

In Table 5 is given a summary of the reference atmospheres used in the various calculations. The original references should be referred to for details. Large differences are apparent in the description of the exposure atmosphere as regards the particle size distribution with which the radon daughters are associated. Also, the assumptions regarding the fraction of unattached daughters are quite different.

The importance of the unattached fraction or the "free ion" component of the exposure atmosphere is that the presence or absence of free ions has a profound effect upon the site and magnitude of deposition in the respiratory tract. The free ions would deposit with virtually 100% efficiency. Lassen and Rau *(56)* and Lassen *(57,58)* gave the rate of attachment (λ_p) of ions to nuclei as:

$$\lambda_p \sim 4\pi r^2 c\nu /(1 + h\nu) \qquad (1)$$

where $\nu = (RT/2\pi M)^{1/2}$ and $h = \nu /D$. In eq. (1), R is the gas constant, T is the absolute temperature, M is the molecular weight of the ions, D is the diffusion constant, r is the radius of the absorbing particle and C is the concentration of the particles per unit volume of air. Croft and Perry *(59)* gave eq. (2) for the fraction of radon daughters present as free ions:

$$f \cong \lambda/(\lambda + \lambda_p) \qquad (2)$$

where λ is the decay constant of the radon daughters.

Table 5. Comparison of the properties of several reference atmospheres.

Reference	Fraction of free ions			Activity on particles <0.1 μm	Diffusion constant		Types of atmosphere	Particle density, cc^{-1}
	RaA	RaB	RaC		Ions	Nuclei		
Altshuler et al. *(46)*	0.09	0.01	0.001	60	0.054	16×10^{-6}	Mine	
Jacobi *(47)*	0.25	0.01	0.002	50		3×10^{-6}	Normal Room air	10^4
Haque and Collinson *(48)*	0.35	0.06	0.08	75	0.054	5.4×10^{-6}	Room air	3×10^4
Walsh *(33)*				100		13×10^{-6}	Mine	$10^4 - 10^5$
Harley and Pasternak *(49)*	0.04	0.002		20	0.054	1.2×10^{-6}	Mine	10^4

The values given in Table 5 are calculated from eq. (1) and (2) except for Jacobi *(47)*, who derived the fraction of uncombined radionuclides as a function of aerosol concentration.

From Table 5, one would expect the Haque and Collinson assumptions, other factors being constant, to yield the highest dose values. From Table 4, it can be seen that this is the case.

Limitations in Deposition Models

Two anatomical models have been used in deposition calculations. Altshuler et al. *(46)* and Jacobi *(47)* chose the respiratory tract model of Findeisen *(60)* in its improved form by Landahl *(61)*. A partial description of the Landahl model is given in Table 6. Haque and Collinson *(48)*, Walsh *(33)*, and Harley *(49)* based their calculations on the more extensive model of Weibel *(62)*. Both these models are too simplified. In calculating deposition, each region is assumed to consist of a number of equal diameter tubes in parallel connected in series with the proceeding region and the following region. Other simplifying assumptions such as laminar flow, circular tubes, and equal inspiration and expiration period were also made. It is not surprising, then, that there is room for variance in dose calculations. There is obviously a need for improved deposition models. Raabe et al. *(63)* are developing a Monte Carlo model of the lung based upon anatomical measurements of the relevant parameters. This more realistic model along with improved equations for deposition which include provisions for turbulent flow conditions, entrance effects and pauses during breathing should provide a significant improvement in deposition calculations.

Table 6. Characteristics of the tracheobronchial region of the respiratory tract.[a]

Region	*Number*	Diameter, cm	Length, cm	Surface, cm^2
Mouth	1	2	7	
Pharynx	1	3	3	
Trachea	1	1.6	12	60
Primary bronchioles	2	1	6	38
Secondary bronchioles	12	0.4	3	45
Tertiary bronchioles	100	0.2	1.5	94
Quaternary bronchioles	770	0.15	0.5	180
Terminal bronchioles	5.4×10^4	0.06	0.3	3400

[a] Based upon the Landahl model *(61)*.

Clearance of Deposited Material

The ICRP Task Group on Lung Dynamics report *(64)* contains valuable data on clearance of deposited materials. Clearance of radon daughters has been limited to consideration of clearance due to mucus movement. Such choice assumes that the radon daughters would undergo radioactive decay and thus deliver their dose before a significant clearance by other possible mechanisms. Haque and Collinson adopted information on the velocity of mucus movement due to Casarett *(65)*, Davies *(66)*, Hilding *(67)*, and Proetz *(68)*. The mean values used were 1.25 cm/min for the trachea and main bronchi; 0.25 cm/min for the lobar (secondary) bronchioles; 0.05 cm/min for the segmental (tertiary bronchioles. For the intrasegmental (quarternary) bronchioles and beyond, they assumed that the radon daughter products decayed *in situ*. Altshuler et al. *(46)* used a reference mucus velocity in the trachea of 1.5 cm/min which led to a transit time for the trachea of 8 min. Estimates of transit times in other regions were based on this reference value for the trachea and an assumed daily mucus production of 10 ml/day. The estimate of 10 ml/day mucus production was based upon a single measurement by Hilding *(67)*. The elimination rate of the radon daughter out of a particular region is given by the inverse of the transit time for that region. In addition to different estimates of mucus velocity, Haque and Collinson *(48)* calculated the transit times on the basis of the Weibel model, while Altshuler et al. *(46)* calculated transit times by using the Landahl *(61)* model. The results are given in Table 7.

If the exposure time is long compared to the clearance time from any region of the tracheobronchial tree, the overall transit time would be governed by the longer transit times although movement between different regions of the tracheobronchial tree may be nonuniform and much faster than the mean clearance time. Holleman *(44)*, Aurand et al. *(69)*, Albert and Arnette *(70)*, and Jacobi et al. *(71)* gave mean clearance of 2–4 hr with various fractions of deposited material removed, for portions of the respiratory tract corresponding roughly to the tracheobronchial tree. Holleman et al. *(44)* found removal of radon daughters insignificant for uranium miners. For materials with longer physical half-lives, removal by mucus blanket has been shown to be significant *(44,71)*.

Table 7. Transit times in different sections of the respiratory tract according to Haque and Collinson and Altshuler, Nelson, and Kuschner.

Section	Transit time, min	
	Haque and Collinson	Altshuler et al.
Trachea	9.6	8
Main bronchi	3.8	6
Lobar bronchi	3.04–7.6	11
Segmental bronchi	18–25.4	37
Subsegmental or intrasegmental	Long	82
Terminal	Long	1980

Walsh *(33)* neglected clearance of radon daughters for the above reasons. Because of large uncertainties in the regional dose calculations and the effects of various parameters in the regional deposition, he considered that regional deposition calculations could not be reliably done. More realistic deposition and clearance models might improve the situation. Walsh *(33)* calculated the dose averaged over the bronchial epithelium of the entire tracheobronchial tree and showed that the dose calculated from the available models would agree with this calculation within a factor of 2.

Critical Tissue

The origin of lung cancer is most likely the basal cells of the bronchial epithelium in the more distal segments of the tracheobronchial tree *(72)*. The thickness of material over these basal cells, including the mucus blanket, a serous layer in which the cilia are bathed, the ciliated cells, and goblet cells, is highly variable. Ciliary action and mucus flow can be altered substantially by deposited irritants. Thus it is extremely difficult to estimate the dose received by the basal cells due to the deposition of radon daughters on the mucus blanket. Some of the

basal cells will not be irradiated at all because they will be protected by a thickness of material which exceeds the range of the alpha particles; others will be reached by the RaC′ alpha and not the RaA alpha while still others will be located well within the range of both alpha particles. The reliability with which we may estimate the dose to the basal cells will be determined in the final analysis by how much we can learn about the location of the basal cells below the surface of the bronchial epithelium and about the changes produced by combinations of inhaled aerosols. Presently, we must estimate the maximum dose which the basal cells will receive with a degree of sophistication dictated by the uncertainties involved.

Altshuler et al. *(46)* measured the bronchial epithelium thickness in one subject and estimated that the minimum depth at which the basal cells were located in the segmental bronchioles was 36μm. He estimated the dose to basal cells at this depth to be 24 rads/yr for (his reference atmosphere) mouth breathing and 13 rads/yr for nose breathing. The difference between nose breathing and mouth breathing is probably nil in comparison to other uncertainties. Haque and Collinson *(48)* estimated the dose at the same location to be about four times higher. The dose per working level month (WLM) as calculated by various investigators is given in Table 4.

Recent measurements by Gastineau et al. *(73)* indicate that except for the trachea and main bronchi greater than 67% of the basal cells are within the range of the RaC′ alpha. The RaC′ alphas, as we will show below, contribute about 95% of the dose to the bronchial epithelium for the "typical" uranium mine atmosphere used. The ranges of the 7.68 MeV RaC′ alpha and the 6.00 MeV RaA alpha were determined by Lea *(37)* to be 71 and 47 μm, respectively. The measurements of Gastineau et al. *(73)* show that the thickness of the bronchial epithelium is highly variable. The measurements are given in Figure 1 as percentage of measurements in each 10 μm segment of the bronchial epithelium. The measurements apply to normal bronchial epithelium. These properties of the bronchial epithelium could be changed drastically by the inhalation of various agents.

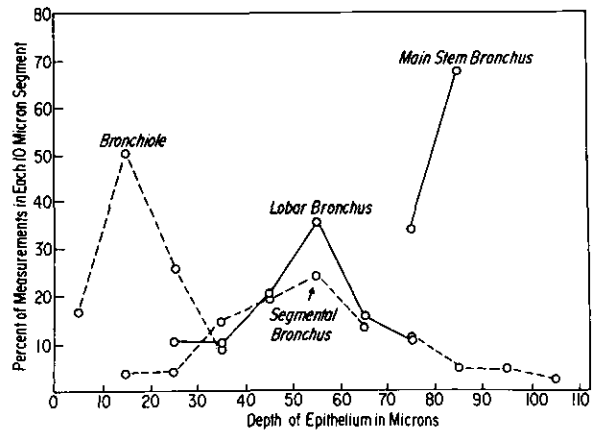

FIGURE 1. Relative percentages of the measurements of epithelial thickness at 10 μm intervals.

Dose Estimate

Altshuler *(46)*, Jacobi *(47)*, Haque and Collinson *(48)*, and Harley *(49)* have calculated depth–dose curves for the RaA and RaC′ alpha in tissue. Although different methods were used (see references for details), large differences were not apparent when the calculations were made independent of deposition calculations. This has been shown by Walsh *(33)* and Holleman *(44)* for the Haque and Collinson *(48)* and Altshuler et al. *(46)* calculations. We may conclude that although different methods of dose calculations were used, the main reasons for the differences displayed in Table 4 are the differences in deposition calculations. Walsh *(33)* has given an approximate mathematical relationship between dose in rads and exposure in WLM. The relationship is given in eq. (3):

$$D_R(\text{rads/WLM}) \sim 0.354\ (f_R v/A_R)\ (S_2 f_{2c}/E_2) \quad (3)$$

where f_R is the fraction of inhaled aerosol deposited in region (see below).

S_2 is the mean stopping power of lung tissue for 7.68 meV RaC′ alpha at depth of penetration of interest, f_{2c} is the fraction of RaC′ alphas emitted at the surface of the region which reach the depth of penetration of interest, A_R is the surface area of the region, $E_2 = 7.68$ meV RaC′ alpha energy, and v is the rate of breathing in liters per hour.

Equation (3) applies if RaA, RaB, and RaC all decay to infinity at their site of deposition.

According to eq. (3), the relative concen-

trations of RaA, RaB, and RaC in the mine atmosphere are not important, because if there is no clearance, most of the dose is due to the RaC' alpha. This is an important consideration, since the relative concentration of RaA, RaB, and RaC in a mine is sensitive to the degree of ventilation of a mine and if the relative concentration were important the working level exposure concept would not be valid. It is important to point out that the fraction of uncombined radon daughters is also dependent upon the degree of ventilation in a mine. The reduction in dose due to reduction of radon daughter concentrations by outside air supply or by air turbulence may be nullified in part by an attendant increase in the fraction of uncombined radon daughters (RaA, RaB, RaC). This is so because a reduction in the concentrations of radon daughters may also tend to decrease the number of available condensation nuclei.

From our previous discussion, the general population is exposed to radon daughter levels which vary from less than 100 pCi/m^3 to 1000 pCi/m^3 depending on geographical location. Since this exposure is for 24 hr/day and 365 days/yr, the WLM to which the general population is exposed is in the range of 0.05 to 0.5 WLM/yr. The estimated dose in rads/WLM varies greatly as given in Table 4. However these estimates which include radon daughters vary from 0.1 to 12 rad/WLM; therefore the dose to the general population could vary from 5 mrad to 6 rad with the dose more likely being between 50 mrad and 500 mrad/yr.

Respiratory Tract Deposition: Plutonium

Sources and Environmental Levels

Plutonium has been distributed over the earth primarily as a result of fallout from atomic weapons testing. Dix and Dobry *(74)* estimate that 300–700 kCi of ^{239}Pu have been released from the testing through 1972. The level of ^{238}Pu is approximately 5% of the ^{239}Pu level or about 25 kCi of ^{238}Pu from fallout. Some of this was released in 1964 when a satellite carrying 17 kCi of ^{238}Pu burned up over the Indian Ocean.

Plutonium levels as measured in shallow water are 0.01 to 0.06 pCi/g with concentrations in marine organism being about 0.7 pCi/kg but the concentration may be as high as 76,000 pCi/kg in bottom-feeding bivalves *(75)*. Air concentrations of ^{239}Pu measured for the period 1964–1966 at Winchester, Massachusetts gave a mean of 0.15 fCi/m^3 *(76)*. In the same study, analysis of human autopsy showed 2.52 pCi/kg of ^{239}Pu in the liver and 1.13 pCi/kg in the lung with relatively little in the rest of the body. Fallout ^{239}Pu is associated with particles of about the same size of that with which other fallout is associated or a mean of about 0.4 μ as noted previously. Shleien *(25)* estimates the dose to the respiratory lymph modes from intake of ^{239}Pu from 1965 to 1968 to be 160 mrem for a 50-yr period and for ^{238}Pu to be 36 mrem.

Plutonium is also produced in significant quantities in nuclear power reactors. A 500 MW(th) reactor operating for 180 days has a fission product inventory which contains about 3.8 kCi of ^{239}Pu *(77)*. In the past, containment of ^{239}Pu has been good with less than a millionth estimated as being released to the environment *(5a)*. Of serious concern is the release of plutonium under accident conditions. As noted previously, the maximum credible accident is visualized as releasing 1–10% of nonvolatiles such as plutonium. However, Eisenbud *(5a)* believes that it is hardly likely that a release greater than 0.1%/day would ever be realized. He also believes that a realistic particle size for the released material is 1 μm.

A more likely prospect of release to the environment will be during fuel reprocessing or during shipment to the fuel reprocessing center. Martin *(78)* in a study of the environmental radiation released by a fuel reprocessing plant estimated 0.06 Ci of gross alpha emitters (including ^{239}Pu) was released to water by a plant which processed 68.8 tons of reactor fuel. Spinrad *(79)* has estimated that about 5,000 gigawatts (GW) of electrical power will be generated by nuclear fission by the year 2000. Using Martin's *(78)* assumption that 30 tons of light water reactor fuel (0.85% ^{235}U) will produce 1 GW of electricity for a year, then 1.5×10^4 tons of fuel/yr will need reprocessing by the year 2000. Therefore based on Martin's study, the release of alpha emitters can be restricted to a few curies per year worldwide under normal operating conditions. Langham *(80)* has estimated the annual production rate of ^{239}Pu to increase from 20,000 kg (1,230 kCi) in 1970–1980 to 80,000 kg (4,960 kCi) by 1990–2000. He estimates ^{238}Pu production will increase during

the same time periods from 10–20 kg (0.6–1.2 kCi) to 6000 kg (360 kCi). He predicts that most of the ^{238}Pu production will be used as power sources in mechanical heart pumps. ^{238}Pu is now serving as heat sources on Apollo stations in the moon and in orbiting satelites. An estimated 0.5 g (8.7 Ci) of ^{238}Pu oxide will be needed to power heart pacemakers and up to 54 g (915 Ci) for the mechanical heart pump. These units will need to be made very rugged to prevent release of plutonium when the users are involved in accidents.

Levels in Human Tissue

Lagerquist et al. *(81)* have reported autopsy results of 19 workers occupationally exposed to plutonium. The average plutonium concentrations (below background to 2 pCi/g) in the lung and trachethial lymph nodes were an order of magnitude greater than in other body organs. This excludes three cases where it was known that exposure to aerosols of plutonium oxide had occurred. The maximum lung concentration was found to be about 44 pCi/g.

Comparison to Radon Daughters

Since plutonium is an alpha emitter, it perhaps seems that the radiation dose results obtained for radon daughters may with a few modifications be applied to plutonium. However, the cases are in fact very different. As noted previously, radon daughters are attached to existing dust particles in the atmosphere with the activity per particle being relatively small (order of 10^{-5} pCi/particle) based on 100 pCi/l. (1 WL) and 10^4 particles/cm^3. This is the same as 1 μCi/10^{11} particles. These numbers were chosen as representative from the previously presented material (see Table 5). Plutonium particles are in most accident type situations made of essentially pure plutonium or plutonium oxide, although in certain situations the plutonium may be attached to other particles in the atmosphere. The particle size will depend on the particular operation leading to the release. Since the activity per particle will be greater for the pure material than for the attached material, the dose evaluation is changed considerably. It is perhaps no longer reasonable to assume uniform deposition over a particular region of the lung when only a few inhaled particles may represent a large fraction of the maximum permissible lung burden. For example, an aerosol of 10 μm diameter ^{239}Pu spheres will give an activity of 1 μCi/1.77 $\times$ 10^3 particles. Since the activity by ^{238}Pu is much higher than that of ^{239}Pu, even less particles are required for a total activity of 1 μCi (about 10 of the 10 μm spheres). This comparison shows that even ^{239}Pu and ^{238}Pu may be expected to give different biological actions, even though both are alpha emitters of about the same energy. It is obvious when the entire lung burden may be represented by less than 10 particles the model of a uniform distribution poorly represents the facts.

The above example is intended primarily to illustrate the extremes of nonuniform deposition and may be representative of very few practicle situations. For a particle of 10 μm diameter, the activity median aerodynamic diameter (AMAD) would be about 30–40 μm so that even if these particles were inhaled, they would most likely be deposited in the upper respiratory tract.

Certainly the particle size will depend greatly on the particular circumstances leading to its production and in accident situations may be quite variable as opposed to the situation with radon daughters where mine atmospheres have received considerable attention. Several reported studies illustrate the expected range in particle sizes. A fire in a plutonium fabrication plant in Colorado involved 400 people, 25 of whom were found to have enough plutonium in their lungs to deliver 15 rem/yr or more *(82)*. The estimated average particle size was 0.32 μm AMAD. This exposure resulted from a chip of plutonium catching fire at a lathe. Even this small amount of material gave an air filter concentration near the source greater than 1 Ci/m^3 and illustrates the severe problems which can arise when fire is involved. Heid and Jech *(83)* reported on 25 inhalation cases involving ^{239}Pu oxide. The AMAD varied from 0.5 to 10 μm with most of the particles around 1–2 μm. For 1.5 μm (AMAD), McClellan *(84)* gives 5.83 $\times$ 10^7 particles/μCi for plutonium oxide. This number may be compared with the 10^{11} particles/μCi arrived at for radon daughters. Further comparison may be made by considering the number of particles per unit area that would be deposited in the lung if completely uniform

deposition is assumed. Hasleton *(85)* has determined that the average adult lung has a surface area of about 40 m^2. If 10^{11} particles are spread uniformly over this area, a density of 2.5×10^3 particles/mm^2 results. The alpha particles emitted by a particle will cover an area of radiation with a radius of about 40 μm in tissue (5×10^{-3} mm^2). Therefore, for a uniform deposition of 10^{11} particles, the areas of radiation would be overlapping with slightly greater than 10 particles being deposited for each 5×10^{-3} mm^2 radiation area. However for the deposition of 5.83×10^7 particles, less than 1 in a hundred of the radiation areas will contain a particle. This means that even in the most uniform deposition that can be imagined that large areas of the lung will not be irradiated. This calculation is made for plutonium that is fixed at a particular location. Clearance, processes and movement of particles will increase the potential number of cells exposed to the radiation.

The radiation dose may be calculated to a 40 μm thick epithelial lining as with radon daughters or to some other "critical tissue" of choice but no longer will there be a relatively high probability that most of the cells in that critical area will be exposed to ionizing radiation. Rather, there will be a few cells exposed to relatively high radiation levels, a few cells surrounding these cells exposed to lower radiation levels and the majority of the cells exposed to no radiation at all. The present methods of calculating maximum permissible organ burdens does not adequately take this into account, especially if the particles are fixed in highly localized areas.

Problems in Dose Estimation

The rem dose is given by

$$D_{\text{rem}} = D_{\text{rad}}(\text{QF})\ (\text{DF})\ (\text{OMF})$$

where QF is the quality factor (a function of the linear energy transfer), DF is the distribution factor, and OMF denotes other modifying factors. The distribution factor can account for some nonuniformity in exposure but is primarily intended to adjust for a nonuniform distribution over the entire organ as occurs when most of the radiation is deposited in the trachea or some other portion of the lung. It is not well adapted to account for a sparse distribution of radioactive particles which must be accounted for by the OMF.

It is of course possible to calculate the dose delivered to the particular tract of tissue through which the alpha particle passes. For example, assume a 5 MeV alpha particle and a 40 μm path that is 0.02 μm wide *(86)*. It is found that 7×10^6 rads is delivered to this tract of tissue for one alpha particle. However, this number alone is of little value.

Those cells exposed to the very high dose levels will be killed. The cells of perhaps greatest concern will be those adjacent to the killed cells which receive only enough radiation to damage them or alter them in such a way that neoplastic tissue develops. For alpha particles which have a short range, the number of these cells for each fixed radioactive particle will be relatively constant regardless of the particle size up to 10 μm. The probability of inducing a cancer may in fact depend as much or more on the number and location of foci as on the total amount of activity inhaled. At present, there is very little experimental evidence to establish how these various factors interact. Experiments are currently in progress *(87)* to study these factors.

Toxicity and Related Factors

In order to assess properly the hazard associated with the inhalation of materials such as plutonium, experimental and epidemiological data with humans are needed. However as Sanders *(88)* observes, "except for epidemiological studies of lung cancer in uranium and flurospar miners there are no data relating absorbed radiation dose from inhaled radioactive materials and tumor incidence in the human lung."

In spite of the paucity of human data, the toxic properties of plutonium have been well established in animal studies. Sanders in his review states that inhalation studies with animals show carcinoma, neoplasm, and tumor induction and notes that the Russian literature reports increased tumor incidence down to 4 nCi or a total radiation dose of 42 rad. He reports a 50% tumor incidence at 3000 rad. Local injections of plutonium oxide leading to neoplasm in

dogs and other studies has led Sanders to conclude that the more localized the plutonium deposition, the more likely a neoplastic change. This conclusion is made plausable when cell proliferation is considered. In areas of heavy plutonium concentration many cells will be killed leading to increased cellular activity to replace the killed cells. Actively dividing cells are known to exhibit the effects of radiation much more readily than nondividing cells *(89)*.

One of the most important factors associated with the toxicity of plutonium which causes it to differ greatly from radon daughters is its long physical and biological half-life (^{238}Pu and ^{239}Pu). The physical half-life has been noted in Table 2. The biological half-life depends on where the material is deposited in the lung, its solubility and the various routes of clearance.

Plutonium's high toxicity may be explained in part by its chemical properties. Plutonium has a principal valence state of +4 and a comparatively small ionic size with high ionic potential with the result that plutonium is a very avid complex former *(90)*. The ability of plutonium to form complexes is greater than in the other transuranics, and insoluble hydroxide ($K_{sp} = 7 \times 10^{-56}$) as well as polymeric hydroxide is formed. This ability may partially account for the observed tendency to aggregate in small regions of the lung *(91)* and the long clearance times. In beagle dogs *(92)*, 85% of the material deposited in the lower respiratory tract or alveoli was retained after 9–10 yr. The ICRP Task Group assumes 90% retention of pulmonary deposits is permanent although some recent work suggest 10% permanent retention and that the other 90% has a long biological half-life of 1000 days *(93)*. Two exposure incidents involving soluble and insoluble plutonium have allowed half-life determinations in the human body *(94)*. For the soluble case, a half-life of 30 days was estimated but for the insoluble case, the estimate was 290 days. These incidents demonstrate the great effect of insolubility on lung retention time. These estimates were for total plutonium inhaled and were not adjusted for the exact location of deposition. Extreme lung retention times have been shown for ^{60}Co in the human of up to 17 yr *(95)*.

Once plutonium has been deposited in the lower respiratory tract, much of it is transported to the respiratory lymph nodes. Sanders *(88)* visualizes the process based on animal studies as follows. The deposited plutonium is rapidly engulfed by lung macrophages which then exhibit reduced clearance action due to the cytocidal action of the plutonium on the macrophages. For example the clearance of FeO_2 is reduced 65% after inhalation of 10 nCi/g lung of ^{239}Pu oxide. The immobilized plutonium-laden macrophages are then engulfed by the alveolar epilthelium, and the plutonium is transported into the pulmonary lymphatic system where sequestration of the plutonium in fibrotic zones of pulmonary tissue occur. The movement occurs on a time scale of years, with less than 10% being transported after the first year but greater than 50% after 5 yr.

The major clearance mechanism from alveoli is apparently by passage of alveolar macrophages to the ciliated epithelium. Sanders *(88)* reports that up to 80% of the macrophages were found to contain plutonium, but 85% of the plutonium activity was on 2% of the sampled macrophages, indicating highly localized deposition even in the alveoli.

From beagle dog studies, Park et al. *(92)* estimate that of the initial amount of plutonium deposited in the alveoli, 10% is retained in the lungs, 40–50% goes to the liver, 5% to the skeleton, and 5% to the abdominal lymph nodes.

The maximum permissible lung burden is 16 nCi (based on 0.3 rem/wk to a standard lung of 1000 g). However, if the lymph nodes are considered the critical organ, then Myers *(96)* finds the maximum permissible organ burden is 0.23 nCi.

A follow-up study *(81)* of 25 plutonium workers during World War II was recently reported 27 years after the group was first exposed to plutonium. The current body burdens were found to range from 0.005 to 0.42 μCi. The lung burdens appeared to be less than 0.01 μCi in all cases. These small body and lung burdens are on the order of the maximum permissible levels but to date none of the medical findings in the group can be attributed definitely to internally deposited plutonium. The particle sizes and initial concentrations of plutonium to which the workers were exposed and where the material was deposited in the lung are not

known and were not given so that complete dose estimates cannot be made. This study, however, indicates that the maximum permissible organ burden for plutonium is a relatively safe number. For this number, which was arrived at on the basis of radiation to the whole organ, it is remarkable and fortuitous that it apparently is still applicable to radiation doses produced by widely dispersed particles.

Reactor Accident: Population Exposure

A light water reactor may have on the order of 100 tons of fuel containing 9–12 kg of plutonium per ton of uranium *(78)* or a total inventory of approximately 1000 kg of plutonium. To illustrate the magnitude of the problem which could arise assume 1% of the plutonium inventory is released as an aerosol over a period of one day due to an accident. One approach to estimating the hazard associated with the release of 10 kg of plutonium is to calculate the possible number of people who could receive a maximum permissible lung burden (16 nCi) from this amount of plutonium. This calculation shows that greater than 30×10^9 people could receive a maximum permissible lung burden. However this calculation is highly unrealistic in that it assumes that the human lung acts as a sink for all released plutonium. A more realistic approach is to assume that this 10 kg (613 Ci) is uniformly distributed in a cloud. In order to estimate the size of the cloud, we assume further that the concentration in the cloud is such that a person immersed in the cloud for 24 hr would deposit 16 nCi of ^{239}Pu in his lungs. Assuming that an adult breathes 20×10^3 l. of air/day and that there is 100% retention of the inhaled plutonium, then the plutonium would be distributed in a cloud of volume 76.5×10^{10} m^3. This equivalent to a cylindrical cloud 1 km high and with a 15.6 km radius. If an inversion with little air mixing were to occur, it is possible the height of the cloud could be restricted to 100 m, in which case the radius would be about 50 km; this could potentially expose many people in a densely populated area. The above calculation and assumption are rather crude, since particle size, time of suspension of the particles, and their distribution in the cloud all depend on the type of accident and local weather conditions. However, the serious nature of the plutonium problem under accident conditions is quite clear, and this problem must be considered in addition to those problems produced by the release of such short-lived isotopes as ^{131}I. It is easily conceivable that a major reactor accident would involve a million people or more, many of whom would receive at least a maximum permissible lung burden of plutonium.

Summary

In this review, we have examined the sources of radiation exposure to the lung of man. Both natural and manmade sources were discussed, and an attempt was made to identify the more important sources. Special emphasis was given to radon and radon daughters and to plutonium. The very different problems presented by these two cases emphasize the importance of understanding the nature of the aerosol and the deposition pattern of any isotope as well as its radiological properties. Even if this information is known, more experimental data are needed on the biological effect as a function of localized versus uniform deposition before an accurate assessment of the hazard can be made.

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